COMMONWEALTH OF MASSACHUSETTS
Executive Office of Health and Human Services

Department of Mental Health
Marylou Sudders, Commissioner

CONFERENCE ON:

Affective Disorders: State-of-the-Art Review

John F. Kennedy Library
September 19-20, 1996

Affective Disorders: State-of-the-Art Review

Sponsored by:
Commonwealth of Massachusetts
Department of Mental Health
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The Commonwealth Research Center, Harvard Medical School;
The Center for Psychological and Forensic Services Research,
The University of Massachusetts Medical Center

in collaboration with

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Massachusetts Chapter, National Association of Social Workers
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Under the Direction of:

William Fisher, PhD
Raymond B. Flannery, Jr., PhD
Shervert H. Frazier, MD
Robert M. Goisman, MD
Alan Green, MD
Lawrence E. Lifson, MD
William Pollock, PhD

Thursday and Friday
September 19-20, 1996
John F. Kennedy Library
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AFFECTIVE DISORDERS & SERIOUS MENTAL ILLNESS:

A STATE-OF-THE-ART REVIEW

THURSDAY, SEPTEMBER 19, 1996

AFFECTIVE DISORDERS: AN OVERVIEW

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Lenore Pollen, LICSW, Moderator

8:30am - 9:00am	***Registration & Continental Breakfast***
9:00am - 9:30am	***Welcome & Opening Remarks*** *Paul J. Barreira, M.D.* *Deputy Commissioner,* *Clinical & Professional Services* *Department of Mental Health* *Raymond B. Flannery, Jr., Ph.D.* *Director of Training* *Department of Mental Health*
9:15am - 10:45am	***Affective Disorders: A Review of the Literature*** *Carl Salzman, M.D.*
10:45am - 11:00am	***Coffee Break***
11:00am - 12:00N	***The Neurobiology of Affective Disorders*** *Alan Green, M.D.*
12:00N - 1:00pm	***Luncheon***
1:00pm - 2:30pm	***The Pharmacological Treatment of Affective Disorders*** *Russell Vasile, M.D.*
2:45pm - 3:45pm	***Psychosocial Interventions with Affective Disorders: Panel Discussion***

1. Psychoanalytic	*Roberta Apfel, M.D., M.P.H.*
2. Cognitive-Behavioral	*Robert M. Goisman, M.D.*
3. Rehabilitation	*David Starkey, Ph.D.*

AFFECTIVE DISORDERS & SERIOUS MENTAL ILLNESS:

A STATE-OF-THE-ART REVIEW

FRIDAY, SEPTEMBER 20, 1996

AFFECTIVE DISORDERS: SPECIAL ISSUES

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Nan Stromberg, RN, CS, Moderator

7:45am - 8:15am	**Registration & Continental Breakfast**
8:15am - 8:30am	**Welcome & Opening Remarks** Alan Brown, M.D., Acting Chair, Department of Psychiatry, University of Massachusetts Medical Center Joseph Coyle, M.D., Chair, Consolidated Department of Psychiatry, Harvard Medical School
8:30am - 9:15am	**Affective Disorders: From Depression to Sadness in Women's Psychotherapy** Irene P. Stiver, Ph.D.
9:15am - 10:15am	**Affective Disorders: Multicultural Issues** Kermit Crawford, Ph.D.
10:15am - 10:30am	**Coffee Break**
10:30am - 11:45am	**Affective Disorders: Issues Across the Life Span** Libby Zimmerman, Ph.D., L.I.C.S.W. Gary Moak, M.D.
11:45am - 12:45pm	**Luncheon**
12:45pm - 1:30pm	**Affective Disorders: Consumers & Families Speak** Evelyn Barkin, Dennis Hagler, Steven Bruhn
1:30pm - 2:15pm	**Affective Disorders: Relapse Prevention** Andrew Nierenberg, M.D.
2:15pm - 2:30pm	**Coffee Break**
2:30pm - 3:30pm	**Affective Disorders: Suicide Assessment** John T. Maltsberger, M.D.
3:30pm	**Conference Closes** (Courtesy Tour of the Kennedy Library)

THE FACULTY

Roberta Apfel, M.D., M.P.H, is Senior Supervisor, Department of Psychiatry, The Cambridge Hospital, and Associate Clinical Professor of Psychiatry, Harvard Medical School.

Evelyn Barkin is a Consumer Advocate, Mental Health Consumer Resource Center, McLean Hospital.

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Alan P. Brown, M.D. is Acting Chair, Department of Psychiatry, University of Massachusetts Medical Center, and Associate Professor of Clinical Psychiatry, University of Massachusetts Medical Center.

Steven Bruhn is a Family Advocate.

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David Starkey, Ph.D., is Clinical Director, Massachusetts Department of Mental Health Metro-South Area, and Instructor in Psychiatry, Harvard Medical School.

Irene P. Stiver, Ph.D., is Visiting Scholar, Stone Center for Developmental Services and Studies, Wellesley College, and Lecturer, Department of Psychiatry, Harvard Medical School.

Nan Stromberg, R.N., C.S., is Psychiatric Clinical Nurse Specialist in Quality Management, Solomon Carter Fuller Mental Health Center, and private practice.

Russell Vasile, M.D., is Director, Out-Patient Psychiatry, Deaconess Hospital, and Assistant Professor of Psychiatry, Harvard Medical School.

Libby Zimmerman, Ph.D., L.I.C.S.W., is Co-Director of Focus Counseling and Consultation, Inc., and Assistant Professor, Boston University School of Social Work.

Statement of Potential Conflict of Interest

Harvard Medical School has long held the standard that its continuing medical education programs be free of commercial bias.

Now, in accord with the potential conflict of interest policy of the Medical School as well as guidelines set forth by the Accreditation Council of Continuing Medical Education and the AMA, speakers have been asked to disclose any personal relationship they have to companies producing pharmaceuticals, medical equipment, prostheses, etc., that might have relevance to the content of their lectures. Such disclosure is not intended to suggest or condone bias in any presentation, but is elicited to provide registrants with information that might be of potential importance to their evaluation of a given talk.

Alan Green, M.D., has received research grants from the following pharmaceutical companies: Janssen, Otsuka, Pfizer, Eli Lilly, Sandoz, and Zeueca. Dr. Green has received honoraria from Lilly and Sandoz pharmaceuticals.

Andrew Nierenberg, M.D., has received honoraria from Eli Lilly, Wyeth-Ayerst, Bristol-Myers, and Pfizer pharmaceuticals.

No other speaker for this conference has reported the potential for receiving something of value from a company whose product may have relevance to the content of this presentation.

ABSTRACTS OF THE LECTURES

AFFECTIVE DISORDERS

Carl Salzman, M.D.

I. Definitions

- A. Unipolar depression
- B. Bipolar depression
  - 1. Bipolar I (mania predominates)
  - 2. Bipolar II (depression predominates)
  - 3. Bipolar III (mania precipitated by drugs)
- C. Delusional depression: delusions of worthlessness, physical illness, paranoid intrusion
- D. Dysthymia: Less severe state with anxiety; present for six months
- E. Atypical depression
  - 1. Anergia, anhedonia, led-pipe paralysis, rejection sensitivity
  - 2. Overlap with personality disorder

II. Epidemiology

- A. Lifetime prevalence 5 percent, Twice as common in women
- B. Recurrent illness: depression increases risk of a second by 50 percent; a second depression increases risk of a third by 75 percent
- C. Comorbidity: anxiety-related disorders, especially panic disorder; medical disorders, especially endocrine disorders and neurologic disorders.

III. Course of Depressive Illness

- A. Risk of recurrence: highest first four to six weeks after recovery
  - 1. Risk persists even after five years following recovery
  - 2. Risks for recurrence include more than three prior depressions, inter-episode dysthymia, strong family history, substance abuse

IV. Etiology of Depression

- A. Psychodynamic theories
  - 1. Early childhood loss + loss in adult life: function; ego ideal; object relations; loss of status hierarchy, dominance, family relations;
- B. Neurobiologic theories: norepinephrine; serotonin

V. Epidemiology of Bipolar Disorder

- A. Prevalence: equal gender; one percent prevalence
- B. Course: recurrent illness; interval between episodes

decreases with age and severity of illness increases with age

VI. Theories of Mania

A. Psychodynamic:
    Shared theories of depression and pathologic narcissism

B. Neurobiologic theories:
    Norepinephrine; dopamine; serotonin

C. Kindling theories:
    Recurrent episodes similar to "kindling" of neuron-firing patterns in bursts with decreased intervals between bursts until there is sustained firing

VII. Clinical Description

A. Bipolar I
    1. Mania. A spectrum from hyperthymia to delirious mania
    2. Dysphoric versus euphoric mania
    3. Mixed manic states

B. 1. Bipolar II

Depression and agitated versus retarded in bipolar I. Hypomania may be difficult to discriminate from normal positive affect.

C. Distinguishing psychotic mania from schizophrenia
    1. Associations
        a. loose versus tangential
        b. flight of ideas
        c. press of speech
    2. Social interaction patterns
        withdrawn versus intrusive
    3. Impaired interpersonal judgement
    4. Absence of negative symptoms
    5. Bizarre versus grandiose thoughts
    6. Malevolent versus grandiose paranoid delusions

D. Dysphoric mania.

    1. Episodes are characterized my irritability, anger, intrusiveness, with little or no euphoria.

    2. Dysphoric mania is more characteristic of older patients in the later stages of the disorder compared with younger patients.

E. Mixed mania.

1. Patients are euphoric and dysphoric alternatively, or mixed together.
2. Tends to be characteristic of severely ill patients.

VIII. Course of Bipolar Illness

A. Bipolar illness is a recurrent disorder.

B. Cycle length may vary.

1. Typically a first episode will occur in adolescence and not be recognized. The second episode my not appear for 5 to 10 years.
2. Episodes do not follow in a predictable pattern. A depression is not always followed by a mania and vice versa.
3. Episodes may be treated by external stress, or may arise without precipitants.

C. Over the course of the illness, episodes become more frequent.

1. The severity of the episodes (both highs and lows) increases.
2. The interval in between episodes shortens.

Neurobiology of Affective Disorders

Alan I. Green, M.D.

The neurobiology of the affective disorders has been an active area of scientific investigation since the introduction of the first clinically effective antidepressant drugs -- imipramine and the monoamine oxidase inhibitors -- in the late 1950's. In the years since then, these first antidepressant drugs, as well as the newer ones as they have come along, have themselves become major research tools. Research into their mechanisms of action has provided the basis for various working hypotheses about the biochemistry of depressions and has also led to more fundamental discoveries about the neurobiology of the central nervous system itself.

The brain is known to contain billions of neurons, each one interacting with others by electrochemical means. When a neuron is stimulated, the resulting impulse, or electrical action potential, causes a release of a chemical substance (a neurotransmitter) from a specialized region close to a neighboring neuron. The neurotransmitter is released into a space between the two neurons, called a synaptic cleft, and then interacts with a neighboring neuron at a receptor site. This interaction may produce electrical stimulation (or inhibition) of the neighboring neuron.

Many different substances act as transmitters in the brain, and many other chemicals can be regulators or modulators of this process. Pharmacological agents, such as the antidepressants, or environmental stimuli of many kinds, ultimately exert their effects by altering neurotransmitter-mediated or neuromodulator-mediated interactions between neurons.

The catecholamine hypothesis and beyond:

The first two classes of antidepressant drugs -- the monoamine oxidase inhibitors and the tricyclic antidepressants -- were introduced into psychiatry about 40 years ago. Within a few years after their introduction, several lines of evidence began to suggest that these medications worked at least in part through effects on catecholamines (norepinephrine, epinephrine and dopamine) or indoleamines (such as serotonin), two groups of neurotransmitters in the brain. One of the neurotransmitters, norepinephrine, seemed to have particular importance in this regard. It was recognized that drugs that treated depression increased the availability of norepinephrine and drugs (such as the antihypertensive agent, reserpine) that depleted norepinephrine caused depression.

Based on these data, the "catecholamine hypothesis of affective disorders" was formulated in the mid-1960's by Schildkraut. This hypothesis proposed, essentially, that some depressive disorders were associated with a relative deficiency of catecholamines, whereas manias were associated with an excess of such catecholamines.

This hypothesis, which has been amplified and expanded over the past 4 decades, remains the foundation upon which our understanding of the neurobiology of the depressive disorders rests. This lecture will describe how our understanding of these

disorders has grown from the catecholamine hypothesis to include information about the action of many neurotransmitters, including serotonin, dopamine, acetylcholine, and gamma aminobutyric acid. Moreover, our new theories about the depressive disorders include information about the effects of these neurotransmitters on their receptor sites in the brain.

Neuroendocrinology:

Because many endocrine (hormone) disorders present with psychiatric symptoms (particularly affective symptoms), investigators have long considered the possible connection between the endocrine system and mood disorders. The discovery that proteins from an area of the brain known as the hypothalamus regulate the release of hormones into the blood stream, prompted further interest in this relationship. In recent years, new techniques have allowed investigators to carefully assess the interaction of the endocrine system and psychiatric symptomatology.

The largest body of information is known about the system that regulates the circulation of the hormone cortisol (produced by the adrenal gland) in people with depression. It has been clear for at least 20 years that those with depression have a dysregulation of this endocrine system. In addition, abnormalities within the regulation of thyroid hormone have also been observed. This lecture will review this information and demonstrate how investigators are seeking clues within abnormal hormonal regulation for information about the underlying neurobiology of the mood disorders.

Psychoimmunology:

Although there has been great interest in the relationship of depression to overall immune function, the findings in this important area have been diverse and often inconsistent. This lecture will include a brief review of our current understanding of this topic.

Sleep in depression:

Depressive disorders are often associated with abnormalities of sleep including changes in time of onset (and intensity) of REM (rapid eye movement) sleep. The relationship of the findings in this area to the biochemical and neuroendocrine abnormalities in patients with depressive disorders will be discussed.

Biological rhythms:

The study of biological rhythms involves an understanding of the daily (circadian) and seasonal changes in neurobiologic functioning. These rhythms, which occur in all animals, can be abnormal in people with depressive disorders. Circadian rhythms of

sleep and hormones can be altered, as can the effects of seasonal changes in light. The role of altered rhythms in patients with mood disorders will be reviewed.

Summary:

This lecture will provide an overview of the current theories of the neurobiology of the mood disorders, as well as an integration of the findings into a theoretical framework wherever possible. It will demonstrate how research in this field is actively evolving through period of accumulation of new information. Moreover, the role of biological findings in the classification of "subtypes" of depressions will be discussed.

Suggested references:

Bauer MS, Whybrow PC. Rapid cycling bipolar affective disorder: II. Treatment of refractor rapid cycling with high dose levothyroxine. A preliminary study. Arch. Gen. Psych. 47:435. 1990.

Bloom F, Kupfer D. Psychopharmacology: The Fourth Generation of Progress. Raven Press, New York, 1994.

Carroll BJ. Dexamethasone suppression test: A review of contemporary confusion. J. Clin. Psychol. 46:13, 1985.

Goodwin FK, Jamison KR. Manic-Depressive Illness. Oxford University Press, New York, 1990.

Green AI, Mooney JJ, Posener JA, Schildkraut JJ. Mood disorders: biochemical aspects. In, Kaplan HI, Sadock BJ (eds): Comprehensive Textbook of Psychiatry VI, Williams and Wilkins, Baltimore, 1995, p. 1089.

Nemeroff CB, Krishnan KRR. Neuroendocrine alterations in psychiatric disorders. In, Nemeroff CB (ed): Neuroendocrinology, CRC, Ann Arbor, MI, 1992.

Schatzberg AF, Rothschild AJ, Langlais PJ, Bird ED, Cole JO. A corticosteroid/dopamine hypothesis of psychotic depression and related states. J. Psychaitric Res. 19:57, 1985.

Schildkraut JJ. The catecholamine hypothesis of affective disorders: A review of supporting evidence. Am. J. Psychiatry 122:509, 1965.

Stein M, Miller AH, Trestman RL. Depression, the immune systemj and health and illness: Findings in search of meaning. Arch. Gen. Psychiatry 48: 171, 1991.

Russell G. Vasile M.D

The Psychopharmacologic Treatment of Affective Disorders

Abstract of Presentation

The objective of this lecture is to provide perspectives on emerging developments in the practice of clinical psychopharmacology relevant to patients with mood disorders. Focus will be placed on the clinical assessment and management of patients with affective illness. A diagnostic overview of affective illness will be discussed, emphasizing major depression, manic-depressive illness and schizoaffective disorders. Subcategories of major depression, including psychotic depression, melancholia and atypical depression will be highlighted, as will rapid cycling bipolar illness.

The lecture will address practical issues in the psychopharmacologic treatment of affective disorders including the choice of specific antidepressants and mood stabilizing medications, and will examine commonly encountered side effects and medication interactions. Medical conditions affecting the choice of an antidepressant or mood stabilizing medication will be described and

strategies for dealing with these issues will be presented. The use of lithium, valproic acid and carbamazepine will be discussed as will the role of clozapine in the treatment of affective illness.

Decision making in approaching the affectively ill patient will be emphasized with a particular emphasis on the question of which antidepressant or mood stabilizing agent to choose in a specific clinical situation. Particularly emphasis will be placed on the selective serotonin antidepressants,.and recently available antidepressants including venlafaxine, nefazodone and fluvoxamine.. The utility of tricyclic antidepressants and other antidepressants including bupropion, amoxapine and trazodone will be described. Additionally, the role of monoamine oxidase inhibitors in the treatment of depression will be explored.

Discussion of the usage of antidepressants in the context of psychotic depression, manic depressive disorder and atypical depression will focus on the unique issues in psychopharmacologic management these disorders present. Factors which impact on the issue of how long and at what dosage a given patient should remain on antidepressants will be reviewed. The subject of the treatment resistant affectively ill patient will be discussed in detail, with an emphasis on alternative strategies including switching between different classes of antidepressants or mood stabilizing medications, and employing combinations of psychopharmacologic agents. Medical conditions which could

be contributing to treatment resistance will be noted. Comorbid psychiatric conditions which frequently present in association with affective illness, such as borderline personality disorder and alcoholism will be discussed in relation to implications for psychopharmacologic treatment planning.

References

1. Vasile RG: "Medical Treatment of Depression " in *Psychiatric Secrets* Edited by Jacobson JL and Jacobson AM. Philadelphia: Hanley and Belfus, 1995

2. Phillips KA and Nirenberg AA: The Assessment and Treatment of Refractory Depression . J Clin Psychiatry 1994; 55:2 (supp): 20-26

3. Finley PR: Selective Serotonin Reuptake Inhibitors: Pharmacologic Profiles and Potential Therapeutic Distinctions. Ann of Pharmacotherapy 1994; 28:1359-1369

4. American Psychiatric Association Practice Guideline for the Treatment of Patients With Bipolar Disorder Am J Psychiatry 1994;151:12 December 1994 Supplement

5. Banov MD, Zarate CA, Tohen M, Scialabba D, Wines JD, Jr. ,Kolbrener M, Kim JW, Cole JO: Clozapine therapy in refractory affective disorders: polarity predicts response in long-term follow-up. J Clin Psychiatry 1994; 55(7):295-300

PANEL

PSYCHODYNAMIC/PSYCHOANALYTIC APPROACHES TO THE TREATMENT OF DEPRESSION

Mary Anne Badaracco, M.D.

1. Depression as a final common pathway of various internal, interpersonal and other environmental factors. Multifaceted etiologies require multifaceted treatment.

2. Psychological Theories of the Etiology of Depression.
   a. Freud Mourning and Melancholia (1917): Loss and anger turned inward; Identification with the lost object.
   b. Karl Abraham: Oral character development predisposed to depression.
   c. Bibring (1953): Discrepancy between ego ideal and actual ideal.
   d. Rochlin (1959): Depression as a superego phenomenon.
   e. Sandler and Joffe (1965): Depression as a basic affect similar to anxiety; necessity of developing defenses to deal with basic depressive affect.
   f. Brown and Harris, The Social Origins of Depression (1973): Depression as a loss of meaning in life.
   g. Brenner, The Mind in Conflict, (1982): Depression is a basic affect and motivates defenses as much as anxiety does; erroneous to think of presence of depression as defining a mental illness.

3. Depression as a Spectrum Phenomenon.

4. Types of Psychological Treatments.
   a. Cognitive
   b. Behavioral
   c. Interpersonal
   d. Psychodynamic/Psychoanalytic

   Types a. through c. were specifically developed for the treatment of depression; Psychodynamic/Psychoanalytic Theory was not.

5. Questions re: Theories
   a. Effectiveness
   b. Differential effectiveness on specific depressive symptoms or in specific psychosocial domains.
   c. Enduring effects beyond current episode.
   d. Advantages in the combination of Psychotherapy and Psychopharmacology.

6. Brief Descriptions of Interpersonal and Psychodynamic/Psychoanalytic

   Interpersonal Therapy: Depression occurs in an interpersonal context. Interpersonal difficulties are a cause and a consequence of depression. A focus on treatment is on current interpersonal issues and not on early developmental ones. The goal of treatment is to master

interpersonal situations without any attempt at personality reconstruction. The techniques include:

1. Clarification (conscious)
2. Encouragement of Affect
3. Communication Analysis

<u>Psychodynamic/Psychoanalytic</u>: Goals are to understand unconscious conflict and defenses. Transference relationship is a key feature of the treatment. Treatment has been less well studied than interpersonal.

7. Some specific relevant studies.

   a. Weissman et al (1974): <u>Treatment Effects on the Social Adjustment of Depressed Patients</u>
   106 patients randomly assigned to Amtriptyline, placebo, and no pill with and without psychotherapy.

   b. Elkin et al (1989): <u>NIMH Treatment of Depression Collaborative Research Program: General Effectiveness of Treatments</u>.
   250 patients randomly assigned to one of four treatment groups for 16 weeks: interpersonal therapy; cognitive therapy; Imipramine plus clinical management; and placebo plus clinical management.

   Results:
   1. All four patient groups had a significant reduction in depressive symptoms and improved functioning.
   2. Significant differences among treatments were present <u>only</u> for the subgroup of severely depressed and functionally impaired patients.
   3. There were <u>no</u> significant differences among the four treatments for the <u>less severely</u> depressed group.
   4. <u>Conclusions</u>: No evidence of greater effectiveness of any <u>one</u> of the therapies as compared with the others, and no evidence that <u>either</u> psychotherapy was less effective than Imipramine plus clinical management for the whole group. But, for the <u>severely depressed</u> (GAS) <u>Imipramine</u> is of value.

8. Psychotherapy of Depressed patients.

   a. A creative process: beware unitary formulations, e.g. Arieti's submission to dominant other or submission to dominant goal.
   b. <u>Consider various factors</u>:
      - Relationship with therapist
      - Acknowledgment of depression and anger
      - Transference/Countertransference issues
      - Establishment of Meaning
      - Exploration of guilt, denial (mania), losses secondary to manic behavior, externalization of responsibility (suicide)

1. Arieti, S., Psychotherapy of severe depression, Am.J. Psychiatry, 134: 864-868, 1977.

2. Bibring, E., The mechanism of depression in Affective Disorders, Ed. By Greenacre P, New York: IUP, 1953.

3. Elkin, I. Et al, National Institute of Mental Health Treatment of Depression Collaborative Research Program: General Effectiveness of Treatments, Arch Gen Psych, 46: 11:971-982, 1989.

4. Karusu, TB., Toward a Clinical Model of Psychotherapy for Depression, I: Systematic Comparison of Three Psychotherapies, Am.J. Psychiatry, 147: 133-147, 1990.

5. Persons, JB, et al, There Role of Psychotherapy in the Treatment of Depression, Arch Gen Psych, 53:283-290, 1996.

6. Shea, MT., et al, Psychotherapeutic Treatment of Depression, in A.P.A. Review of Psychiatry, Vol. 7, Washington, D.C.: A.P.A. Press, 1988.

7. Weissman, et al, Treatment Effects on the Social Adjustment of Depressed Patients, Arch Gen Psych, 30:771-778, 1974.

PANEL

COGNITIVE-BEHAVIORAL APPROACHES TO DEPRESSION

Robert M. Goisman, M.D.

The work of Aaron Beck, David Burns, and others since the 1970's has served to popularize an approach to the treatment of depression which is relatively easy to learn, capable of being delivered within a short-term or managed care environment, and accessible by patients across a wide variety of backgrounds and levels of psychological and socio-economic functioning. Often referred to as "cognitive" or "cognitive-behavioral" therapy, this form of treatment has been found effective in a number of carefully controlled and methodologically sophisticated studies. It can be used alone in milder cases, or it can be combined with medication for more severely or chronically ill patients. It also can be combined with more traditional, psychodynamic treatment, within one therapy or by two therapists working together or sequentially.

In the cognitive-behavioral model of depression, the focus is on how a depressed individual thinks and what he or she does. This does not rule out biological cause of depression, nor does it rule out such traditionally invoked psychological causes as parental loss or unavailability. It focuses on the thinking and behavior which maintain depression and is less concerned with what originally caused it. In Beck's model this thinking is called the Cognitive Triad and consists of a negative view of oneself, a negative interpretation of ongoing events, and a negative view of the future. These views are mediated by schemata, unspoken and rigid assumptions which are "depressing" (e.g., "If I am not loved by others, my life has no value") and through which all incoming stimuli (experiences, memories, etc.) are filtered.

In cognitive therapy, it is the job of the patient and therapist to decipher these schemata, as it appears they have a lot to do with maintaining a depressed state. They can be picked up by monitoring the thoughts which appear in specific situations in which the patient feels particularly or acutely bad. These thoughts, called dysfunctional cognitions or automatic thoughts, tend to fall into certain patterns which can be learned and then searched for. Some of these patterns have become quite well-

known and include"all-or-nothing thinking", "disqualifying the positive", "fortune-telling", and so on.

Typically, although the treatment is referred to as "cognitive-behavioral," the order in which the treatment is carried out is the reverse, i.e. first behavioral and then cognitive, since actions are easier to change than are thoughts. A beginning patient may be asked to make a schedule of his/her day and record his/her mood for each hour. Then might come a Mastery and Pleasure Schedule, in which the patient is asked to record one pleasurable activity and one success experience for each waking hour. A next step might be Graded Task Assignment, in which tasks felt to be overwhelming are broken down into small steps, the completion of each one of which moves the patient progressively closer to the stated goal.

Once this part of the treatment has been successfully negotiated, the focus switches to the thoughts which accompany depressed mood. Here patients are asked to begin recording their thoughts, using a form called the Daily Record of Dysfunctional Thoughts. These forms are filled out each day and brought in to therapy, where it is the job of the patient and the clinician to look for patterns in them, classify these patterns, and then gently begin to dispute them through role-playing, hypothesis-testing, and other techniques. The end-point of therapy is reached when the patient is having significantly fewer dysfunctional thoughts and his/her mood has improved to acceptable levels; the patient at termination should also have demonstrated enough grasp of this method that it can be used by the patient if needed in the absence of the therapist.

Patients most likely to benefit from this approach, with or without antidepressant medication, include those with dysthymic disorder and those with recurrent unipolar major depression. The ability to agree to do written homework is a precondition of successful cognitive-behavioral treatment, and the results of the treatment, like many other human endeavors, are often proportionate to the amount of effort expended.

REFERENCES

1. Beck AT: Cognitive Therapy and the Emotional Disorders. New York: International Universities Press, 1976.

2. Beck AT, Rush AJ, Shaw BF, et al: Cognitive Therapy of Depression. New York: Guilford Press, 1979.

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PANEL

Psychiatric Rehabilitation Integrated Service Model (PRISM): Implications for Individuals with Affective Disorders

David Starkey, Ph.D

I. Psychiatric Rehabilitation Integrated Service Model: An Overview

Despite the current emphasis on community-based treatment and support, many individuals with serious and persistent mental illness remain in state hospital settings. PRISM has been developed to improve the quality of life for patients with serious and persistent mental illness and to increase the possibility of movement to less restrictive settings. This rehabilitation based model differs from those focused upon department or program models in changing the nature of the hospital itself to reinforce patient autonomy and decision making, and to make the entire facility a rehabilitation oriented treatment milieu. This is accomplished by:

- Involving patients in their own treatment by encouraging patient participation in team meetings, and implementing a goal oriented as opposed to a problem oriented treatment plan.
- Providing patients with the supports and skills necessary to advocate for themselves in the hospital setting.
- Involving patients in unit-based and organizational decision-making by forming patient advisory committees and including patients on regular hospital committees.
- Providing skills training opportunities for all patients, regardless of level of functioning (e.g. Social Skills, Symptom Management, Community Reintegration, and Work Skills).
- Involving direct care staff in treatment planning to ensure continuity of care and as a way of reinforcing learning of the PRISM model
- Providing ongoing training for staff in problem solving, conflict resolution, and integrating day to day interactions with the PRISM model

PRISM promotes the formation of structured, nonthreatening relationships between patients and staff - both administrative and clinical. Patients are encouraged and empowered to participate more fully in their own recovery. These principles represent a substantial shift from previous models of treatment for patients at Medfield State Hospital, one third of whom suffer from affective disturbances.

II. Monitoring and Evaluating the Impact of PRISM Initiatives

To monitor and assess outcomes related to PRISM initiatives, a group of fifty patients are being followed for a two year period. The outcomes being measured include quality of life, functional skills, subjective assessments of patient empowerment, and patient satisfaction. Data collection instruments consist of:

- Quality of Life Scale (Lehman)
- Ward Atmosphere Scale (Moos)

- Internal-External Locus of Control Scale (Rotter)
- Expanded Routine Task Inventory (Allen)
- Making Decisions Scale (Rogers)
- Patient Satisfaction and Opinion survey (developed for use with this model)
- Data on demographics and individual patient characteristics

Data collection has taken place at the initiation of the study, and at 6 and 12 month intervals thereafter. The program is currently nearing the end of its second year.

III. PRISM and Affective Disorders: Implications and Analysis

We have hypothesized that the presence of affective symptoms may result in the following:

1. Measures of quality of life, empowerment, locus of control, and functional skills will be higher at baseline for the group of patients with affective symptoms than for those patients without affective symptoms. If this is so, it may be possible to discriminate between the two groups based upon a particular cluster of characteristics and/or symptoms.

2. The interpersonal components of PRISM, such as patient/staff co-led community meetings and patient advisory committees will promote a greater degree of involvement among patients with affective symptoms versus patients without affective symptoms.

3. Increased quality of life, better functional skills, higher subjective levels of empowerment and increased participation in treatment will lead to better outcomes for the group of patients with affective symptoms than for other patients (such as schizophrenic patients without an affective component to their illness). Patients with affective symptoms may benefit from PRISM-related initiatives to a greater degree than patients in other diagnostic categories. An attempt will be made to identify which specific components are more effective for this patient group.

Discriminant analysis, analysis of variance, and nonparametric statistics are being utilized to determine the validity of these hypotheses.

IV. Preliminary Findings

Preliminary analysis of one year data indicates that differences among groups are emerging. Interestingly enough, these differences are not necessarily where we would expect to see them, nor are they in the direction we might anticipate. Differences between groups, as well as what impact those differences might have on patients' ability to benefit from PRISM initiatives, will be discussed during the presentation. In addition, we will discuss the implications of our findings with regards to the design of rehabilitation interventions for people with affective symptoms.

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From Depression to Sadness in Women's Psychotherapy

Irene Pierce Stiver, Ph.D.

Current literature tells us that twice as many women as men undergo depressive episodes; and 1 out of every 10 women can expect to experience a serious depression in her lifetime. Married women are more prone to develop depressive illness than both married men and single women who are heads of households. Traditional theoretical approaches to depression do not adequately take into account this greater incidence of depression in women nor do they help us understand these striking findings.

This presentation will explore why women become depressed and will do so through a focus on sadness; some therapeutic implications of this emphasis will be identified. We believe that significant and qualitative differences exist between sadness and depression, phenomenologically. It is a difference between a "feeling state" (sadness) and a state in which feelings are hidden -- a "non-feeling state" (depression), but with clear dysphoric components. When there is not an adequate relational context in which sadness can be experienced and validated, depressive reactions develop.

The relative lack of attention to "sadness" and its role in depression may occur because sadness is a powerful affect. Intense emotion is often seen as more characteristic of

women's experience than men's. It is both devalued in our culture and threatening to those who are more defended. Our culture admires and values the more stoical responses to grief and devalues more open expressions of sadness.

Our basic notion is that many women who suffer depression have not been able to experience their sadness within a context of empathic and validating relationships. One of the major reasons that this occurs is that the people in the surrounding context of relationships do not recognize that a disappointment or loss has occurred. Not only do they not help the woman acknowledge the loss, they often prevent her from doing so, which contributes to severe confusion and self-doubt.

The most growth enhancing therapeutic encounters are those which provide for mutual experience of connection through the therapist's readiness to sit with the patient's pain. The therapist tries to help her patient identify the underlying sadness and then "bears" with her what appears as unbearably intense affects. It is the very expression of authentic feelings which strengthens the connection between patient and therapist and allows them both to move in a mutually empowering way. The patient then no longer feels alone and bereft. As she feels more understood and as her feelings develop more clarity, she can experience more positive self worth and can begin to hope for and move toward more gratifying connections in the future.

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AFFECTIVE DISORDERS: FACT OR FICTION WITH POPULATIONS OF COLOR

Kermit Crawford, Ph.D.

Does the term Affective Disorder have utility in culturally competent clinical practice, or is it obsolete? There is no diagnostic category in DSMIV designated as Affective Disorders. Perhaps too often the term Affective Disorder is used interchangeably with Mood Disorder, when they are in fact not equivalent terms. Affect is generally defined as observable patterns of behavior that express an individual's subjective feeling or emotional state. Mood is typically defined as the underlying and sustained subjectively experienced emotion. In the commonly used mental status examination, a client's affective presentation can aid the identification and assessment of many mental disorders, including mood disorders, but the two are not the same.

So where does the notion of affective behaviors belong? Is the commonly used notion of affective presentation still relevant to mental status assessment and evaluation? Although affect does not constitute a diagnostic category of mental disorder, it is widely recognized as a mainstay in the assessment of mental disorders. Affective behaviors seem to respond both to internal and external cues. With exception of some Asian-American groups People of Color, defined broadly as the four historically American minority groups of African-American, Latino/a, and Native Americans, and Asian-Americans, typically experience higher prevalences of mental disorders. Among Asians in America, the prevalence of mental disorders is widely acknowledged to be higher than is officially recorded. This has led some researchers to cite the lack of, and need for, a valid large scale epidemiological study of this population (Sue et.al.,1994). Although there appears to be some increased in risk of some mental illnesses among populations of Color, the reality is often not nearly as extreme as some have attempted to make it appear. For instance, prevalence figures have been grossly distorted such as the notion of "mark of oppression" which implicated mental illness simply due to one's status as African-American (Kardiner and Ovesey, 1951). Race, culture and mental illness have often appeared to be "politicized" in this country. This observation has led some theorists to speculate that it may not have been coincidental that the Civil Rights Act of 1964 and 1965 corresponded with the passage of the Community Mental Health Centers Act in 1963 (Turner ad Kramer, 1995).

Does observed affective presentation have the similar meaning across the predominate racial and ethnic groups in this country? Observed affective presentation can have similar meaning, when the clinician has knowledge of culture and ethnicity. A more pertinent question may be whether or not affective presentation is a reliable tool for the assessment of People of Color as it is generally held to be in the assessment of the racial majority individuals in the United States? A quick response may be "Of course". This may be in part related to the sometimes limited state of the technology in which the mental status assessment is embedded. It could relate to a lack of awareness of the implications of reliance on this technique. Or it could relate to a lack of knowledge

of non-traditional diagnoses. For instance, many noted clinicians are not aware of the Culture Bound Syndromes listed in Appendix I of DSM IV. Many of which talk to the affective presentation as key in differential diagnosis. A more reasoned response would be,"when certain conditions of knowledge, skills and awareness are met".

As professional clinicians, we are poked and prodded daily by developments in the field, re-worked technology and the like. We should be focused on the "bells and whistles" in our field, but we should not forget the search for basic knowledge, skills and awareness which too often may elude us. While use of affective presentation may be central to the mental status assessment, there is much learning and work to be done to fully realize the potential of this powerful tool. This presentation will focus on culturally competent clinical assessment technology, with specific focus on the skills, knowledge and awareness aimed at enhancing the utility affective behaviors, and of general clinical presentations, both in research and in practice with People of Color.

AFFECTIVE DISORDER: FACT OR FICTION WITH POPULATIONS OF COLOR

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Conceptualization and Treatment of Depression in Infants, Children and Adolescents

Libby Zimmerman, Ph.D.

We expect children to be lively, engaging and eager to meet others. A child's enthusiasm and delight is often infectious and can bring an adult into mutually rewarding interactions. When children are withdrawn, sad and lacking in energy, adults seem to withdraw and find it hard to face the painful reality. This is manifested in the historical, under-diagnosis of depression in children.

Over the last two decades there has been increasing interest in the early diagnosis and treatment of affective disorders in infants and children. The diagnosis of depression in children and adolescents continues to be merged with the diagnosis of depression in adults in the latest Diagnostic and Statistical Manual. However, a diagnostic classification of mental health and developmental disorders of infancy and early childhood, published in 1994, by the National Center for Clinical Infant Programs supports clinicians and researchers efforts to describe the unique manifestations of depression and other mental health problems in infancy and early childhood.

Since children grow and change rapidly, a developmental approach is central to understanding depression in children. The developmental approach supports the idea that a crucial link exists between the biological make-up of a child and the quality of that child's interaction with caregivers in the context of the social and economic supports available to those caregivers. Although the relational context is important in understanding depression across the life-cycle, we are required to look at reactions of significant caregivers to understand depression in children.

Traditional psychodynamic approaches to mental health focus on the internal organization of the individual and the necessary resolution of a series of internal epigenetic crises to achieve individuation and separation from a loved "object" or primary caregiver. Depression within this framework is seen to result from a failure to separate from the loved "object". Thus the concept of dependence permeates traditional thinking about depression.

Over the past fifteen years direct observations of infants and young children, interviews with depressed women, and new clinical explorations with adults, combined to create an interpersonal approach for understanding the development of the self and of depression. This relational theoretical framework views independence and a strong sense of self as growing out of strong connections with other people.

An interpersonal approach guides us to look at the experience of depressive feelings in the child, or identified patient, and the feelings in the significant people who relate to that person. Within this approach it is possible to understand how the inter-subjective feelings of each person in the relationship influence each other in significant ways. Thus researchers find that adults may respond to children's depressive feelings with depressive feelings of their own. Adults may then feel sad or even helpless. In some instances they recognize those feelings and respond with caring. In other instances they may restrict their feelings, and withdraw from such painful transactions. In either case, a depressed child influences the adult's response and the adult's response influences the child's inter-subjective world. These exchanges are significant and occur with family members, teachers and with clinician-researchers.

Diagnosis of depression in adults rests heavily on self-reports of low self esteem and a sense of rejection. Young children often do not have the vocabulary to share their inner experience in a way parents or teachers understand.Although it is possible to observe some of the symptoms of depression, such as deep sadness, diminished interest and pleasure in people and activities, other symptoms such as low self esteem and a sense of rejection are not so easily observable.

Symptoms of depression such as irritability, mood swings, and withdrawal are often hard to distinguish from normal patterns in child development. The difficulty in diagnosis is confirmed by empirical studies that report psychiatrists find it more difficult to agree on ratings of depression than those of anxiety. Differentiating sadness from depression is a challenge for parents and teachers. Taking action when the diagnosis is clear presents another challenge. Research suggests that parents and teachers frequently fail to take action in response to depression even when older children and adolescents report feeling depressed.

This presentation will present a framework and provide guidelines for assessment and treatment of depression in infants, children and adolescents. The assessment focuses on the visible behavior of infant and caregiver and the inter-subjective world of child and adult. This framework provides support for responding to, rather than withdrawing from, the reality of childhood depression.

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GERIATRIC DEPRESSION
Gary S. Moak, M.D.

According to most psychiatric epidemiologic studies, the prevalence of clinically significant depression among community-residing elderly is about twenty percent. Only five percent of community geriatric samples, however, meet criteria for a diagnosis of Major Depression. The explanations for this discrepancy reveal much about what is different between depression in the elderly and that which affects younger adult populations.

One possible reason that Major Depression is much less prevalent among the elderly even though depression is a much more common problem is that standard diagnostic criteria for depression may not be valid for geriatric depression. An axiom of geriatric medicine is that disease presents atypically in the elderly. Many common disorders of old age, such as myocardial infarction, stroke, and urinary tract infection, present without the classic signs and symptoms by which they usually are recognized and diagnosed. For example, myocardia infarction often presents without chest pain. This phenomenon may be true for depression as well. Elderly depressed patients may present without depressed mood; other dysphoric moods, such as anxiety or irritability, may predominate the clinical pecture. Ideational syptoms, such as rumination of guilt, hopelessness, nihilistic beliefs, or suicidal ideation, may be less evident. On the other hand, somatization, cognitive deficits, and functional impairment may be much more prominant. Threshholds for recognizing changes in social and ocupational functioning may not be sufficiently sensitive for people who are retired, have restricted activities, or live alone or in nursing facilities. Neurovegatative symptoms of depression may be hard to discern in the presense of other illnesses of old age which cause anergia, anorexia, insomnia, fatigueability, and constipation. Moreover, elderly patients are notorious for underreporting symptoms of disease. Thus, a different or more broad set of criteria may be needed by which to diagnose depression in the elderly.

While it seems true that depression often presents differently in old age, it may also be true that some depression in late-life represents a different disease entity than that seen in other age groups. Biochemical changes which occur in the brain during the aging process may affect the vulnerability to depressionas well as the expression of depressive symptomatology. Some researchers speculate that depression may represent a final common pathway for at least one dimention of brain failure in the elderly.

In fact, forty percent or more of geriatric depression is secondary depression- depression caused by another illness. General medical illness and neurologic disease commonly are associated with depression. Many medications prescribed to treat both nonpsychiatric and psychiatric disorders have depression included in their lists of known side effects.

The cause-and-effect relationships between depression and nonpsychiatric illness in the elderly usually are complex. This is especially true for neurologic conditions. Disease such as Alzhiemer's Disease, Vascular Dementia, stroke, and Parkinson's Disease have a prevalence of associated depression in the range of thirty to forty percent. In these diseases, depression may come about as a direct expression of brain dysfunction (i.e., as a neurological symptom). In this scenario, an organic mood disorder would be the appropriate diagnosis. Conversely, comorbid depression may result in an apparent worsening of neurologic symptoms and dysfunction. In this situation, the presence of depression can go unrecognized while the assumption is made that a known neurologic disorder, such as Alzheimer's Disease, has worsened. Finally, the significant personal losses associated with neurologic disease may lead to a depressive psychologic reaction.

The high prevalence of comorbid depression and dementia is an especially difficult challenge in geriatric psychiatry. Because these two neuropsychiatric syndromes have overlapping presentations, it often can be clinically difficult to dissect out their relative contributions. A demented patient who becomes depressed may appear more confused and more abulic. Depression, in such cases, represents an important cause of treatable excess disability that often goes untreated.

The overlap between depression and dementia can present particular difficulties in the evaluation of elderly patients with schizophrenia. Chronically mentally ill patients who grow old with schizophrenia also are vulnerable to age-related disease such as dementia and geriatric depression. After periods of relative stability during mid-life, such patients may experience late-life neurobehavioral decompensation. Among elderly schizophrenic patients, negative symptoms may assume much greater prominance that positive symtoms. When functional decline occurs, it may be very hard to sort out the relative contributions of such negative symtoms from frontal lobe symtoms of dementia (especially if apathy is prominant) and depression.

Treatment of depression usually is more difficult in the elderly than in younger adults. Ongoing diagnostic assessment must be rigorous, especially because antidepressant treatments can lead to delirium. When subtle, the symptoms of delirium in the elderly overlap with both those of depression and dementia. Sensitivity to side effects is much greater as is the likelihood of drug interactions. Moreover, response latency to antidepressant medication is longer. An adequate trial of an antidepressant may require as long as twelve weeks at therapeutic levels. Thus, treatment of depression in the elderly is more painstaking, taking more time but requiring more intensity of service. Notwithstanding these difficulties, response rates are nearly as high as in younger patient populations.

Unfortunately, much depression in the elderly goes untreated. There are several reasons for this, but one common factor is cultural bias about psychopathology and old age. A common falacious believe often held by patients' clinicians and families is that depression is an understandable or expectable reaction to losses and other setbacks of old age. The belief that old age is depressing is belied by sociologic studies which demonstrate good life satisfaction among the elderly, even in the face of significant adversity. Beliefs that depression in old age is inevitable often lead to failure to seek or offer treatment. Considerable professional and public education still is needed to overcome this attitude.

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AFFECTIVE DISORDERS:

CONSUMERS AND FAMILIES SPEAK

Evie Barkin
Dennis Hagler
Steven Bruhn

Affective Disorders impact on consumers and families in a variety of ways. The illness can impact on a person's ability to function in many areas of their life. The ability to learn, work, socialize, and attend to routine tasks of daily living can all be impaired. Successful treatment must therefore attend to the whole person, not just the overt symptoms of their illness.

The impact of the illness is often felt by familiy members as well. A family member's impairments in functioning often have consequences for other members of the family. In addition to the caretaking role they may have to play for their family member, they may also have to take on additional household tasks, family responsibilities, and financial burdens. As the illness is hereditary, there may often be more than one family member who is affected.

Through a discussion of their personal experiences, the panel will address many of the issues that consumers and their families face.

Evie Barkin will discuss how depression affected her life and relationships, and what she did to help herself. She will focus on the importance of being able to hold onto hope.

Dennis Hagler will give a brief summary of the progression of his illness and his road to recovery. He will discuss the importance of medication, and the reasons why people stop taking their medication. He will give treatment providers feedback on what consumers find helpful and not helpful to their recovery process.

Steven Bruhn will relay his personal account of growing up in a family in which several family members developed Affective Disorders. He will discuss the emotional and financial burdens placed on the family, and the frustrations that family members encounter in trying to access treatment when a family member becomes ill.

Panelists hope that by sharing their personal struggles with us, we can develop a better understanding of their needs. All of the panelists are members of the Manic Depressive and Depressive Association - Bipolar and Unipolar Self Help and Support Group.

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a) Observational Studies and Uncontrolled Case Series

Lavori and colleagues followed 359 patients from the NIMH Collaborative Program on the Psychobiology of Depression (1993). These patients had recovered from the index episode for at least 8 weeks and were then followed for up to 5 years. The investigators found that of those patients who continued to take high levels of somatic treatment, 20.8% became depressed again within the first 6 months. Thereafter, patients who had continued to feel well up until that time relapsed at the same rate regardless of treatment status. Overall, 88% of the entire cohort had a relapse or recurrence by the end of 5 years. The clinical implications of these findings are profound: when followed naturalistically, many patients become depressed again despite adequate long-term pharmacotherapy and the advantages of maintenance antidepressant treatment may diminish after 6 months of wellness. Of great concern, patients who develop a recurrence and then recover from the second depressive episode are at risk of developing even more episodes. Furthermore, depressive relapse and recurrence rates may be substantially higher in clinical, as compared to research, populations. Note that this study was done before SSRI antidepressants became available in the United States.

In another observational study, Ramana and colleagues followed 70 patients (53 in- and 17 out-patients) for up to 15 months (Ramana et al. 1995) : 80% remitted by 15 months; of those who remitted, Kaplan-Meier survival analysis showed that 40% experienced at least one depressive relapse within 10 months of remission. Relapsers and non-relapsers had the same levels of long-term antidepressant treatment. Of note, many of these patients were treated with SSRI antidepressants, but the proportion of patients taking SSRIs was not reported.

b) Controlled Studies of Continuation Treatment

More structured information regarding the occurrence of relapses and recurrences during long-term antidepressant treatment can be gleaned from the literature on continuation and maintenance treatment studies. Continuation treatment is given within the first six months after an acute response to antidepressants, during the time at risk of relapse. Maintenance treatment is after six months of continuation treatment, during the time at risk of recurrence. This literature has generally reported that long-term antidepressant treatment is superior to placebo in double-blind placebo-substitution studies. Patients who are switched to placebo have between a 50% and 80% chance of relapse while those who are maintained on active antidepressants have only 20% to 40% relapse rate. If, however, these data are viewed from the vantage point of failure of continuation treatment, about 20% to 40% of depressed patients become depressed again during controlled trials and up to 80% have depressive breakthrough in naturalistic follow up studies. Key studies are discussed below.

Doogan and Caillard (Doogan et al. 1992) followed depressed patients who responded to an eight week trial of sertraline. After patients responded, they were randomized to either continue on the sertraline (N=184) or be switched to placebo (N=105). Thirteen percent of the sertraline patients and 45.7% of the placebo-substituted patients had another episode of major depression within 44 weeks. Note that if a depression reappeared, the dose of sertraline could be increased to up to 200 mg daily (mean daily dose ranged between 69.3 mg and 82.1 mg daily). The authors do not specify if patients who had another depressive episode, and then responded to an increase in dose, were considered to have a relapse or

recurrence. No mention is made of how many patients required, and then responded to, an increase in dose. Furthermore, although about 75% of the original group had recurrent depression, information that describes the number of prior episodes is missing. For these reasons, the investigators' finding that 13% of patients relapsed during long-term sertraline treatment may be an underestimate of the true relapse rate.

Montgomery and Dunbar (Montgomery et al. 1993) treated 172 depressed patients who had a history of recurrences with open paroxetine (20 to 40 mg/day) for 8 weeks. Patients who responded were randomized to either continue on paroxetine (20 to 30 mg/day;N=68) or placebo (N=67) for the next 52 weeks. Three percent of the patients who took paroxetine had depressive relapses as compared to 19% of the placebo group. Similarly, Claghorn and Feighner (Claghorn et al. 1993) followed patients for 52 weeks and found relapse rates of 25% among 32 patients who had responded to placebo initially, 15% among 60 paroxetine-treated patients, and 4% among imipramine-treated patients. One detail of great relevance for the current grant proposal is that the dose of long-term medication used in Montgomery's study could be maintained, decreased, or increased at the discretion of the physician. The mean dose of paroxetine that patients responded to was 40 mg during the acute trial and 38 mg during the extension phase. The dose of the SSRI in this study, therefore, was stable during continuation and maintenance treatment. As with the studies noted above, no information is provided about subsequent treatment of those patients who had breakthrough depressions while on active medications.

c) Uncontrolled Studies of Maintenance Antidepressant Treatment

A large naturalistic follow-up of consecutive patients who had responded to treatment was published by Peselow and colleagues (Peselow et al. 1991). They followed 217 patients with unipolar depression who responded to tricyclic antidepressants (mean imipramine-equivalence dose 159.6 mg daily), were stable for at least 6 months and then followed for up to 5 years. They compared this group with 28 patients who were stable for six months and then decided to discontinue medication. Survival analysis showed that the probability of a recurrence was between 60% and 80% for the medication group and between 85% and 92% for the discontinuation group. As shown above with controlled continuation treatment, medication is better than none at all; the recurrence rate for patients who take maintenance medication, however, is unacceptably high.

d) Controlled Studies of Maintenance Antidepressant Treatment

Only one study has addressed the efficacy of fluoxetine for long term maintenance therapy. Montgomery et al. compared fluoxetine (N=88) with placebo (N=94) to prevent recurrences in unipolar depressed patients who had responded to an open trial of fluoxetine (40 mg to 80 mg daily) (Montgomery et al. 1988). Subjects were stable for six months on fluoxetine 40 mg/day prior to randomization to either staying on the same dose of fluoxetine 40 mg or switching to placebo. At the end of one year, 26% of subjects who took prophylactic fluoxetine and 57% who took placebo had a recurrence. As with the Frank and Kupfer studies, (Frank et al. 1990; Kupfer et al. 1992) Montgomery et al. demonstrated that active antidepressant maintenance therapy is superior to placebo, but that a substantial proportion (26%) of patients experience a depressive recurrence despite active treatment.

One study of the long-term treatment of depression with paroxetine differentiated continuation and maintenance prophylaxis (Montgomery et al. 1993). Paroxetine was superior to placebo, but 14 % of patients experienced a recurrence during active long-term paroxetine treatment.

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Suicide in Affective Disorders

John T. Maltsberger, M. D.

I. History

- A. Emil Kraepelin (1856-1926). *Textbook of Psychiatry (Lehrbuch der Psychiatrie)* first appeared, 1883. Many subsequent editions, but the best English version appeared in 1902, an abridgement of the 6th edition, by A. R. Defendorf. Various translations of Kraepelin are still available (see bibliography)
  - 1. Kraepelin recognized two classes of affective illness:
    - a. Melancholia, which he classified as an "involutional psychosis".
    - b. Manic-depressive insanity, divided into
      - (1) maniacal states: hypomania, mania, and delirious mania
      - (2) depressive states: simple retardation, retardation with delusions and hallucinations, and stuporous conditions
      - (3) mixed states: maniacal stupor, and stuporous mania.
- B. Freud, Sigmund (1853-1939). Most important contribution was "Mourning and Melancholia," but the 1910 Vienna symposium on suicide in the young was of major importance.
- C. After World War II. psychiatric nosology remained confused, but under the influence of Eli Robins at Washington University, St. Louis, the psychiatric nomenclature was revised with the appearance of *DSM-III* in 1980

II. Current nomeclature, *DSM-IV*, recognizes about 19 different syndromes as affective disorders, with melancholia relegated to an appendix. Suicide is intimately associated with depression, and not only in affective disorders, but in other Axis I. diagnoses.

III. Suicide overview: Robins, *The Final Months*. Reviewed studies by Barraclough and Dorpat & Ripley, and reported on 134 consecutive suicides in St. Louis. Conclusions:

- A. Suicide is unusual in the absence of an Axis I. diagnosis.

B. In any consecutive series of suicides, the commonest diagnosis is major depressive illness, which accounts for between 30 and 70 percent of deaths.

C. Other common diagnoses are alcoholism and schizophrenia. In Robins's study, the patients with depression for a diagnosis when combined with those with a diagnosis of alcoholism accounted for 72 percent of all deaths.

IV. Depression as defined in *DSM IV* in insufficient to characterize suicidal patients.

A. Diagnosis of a major depressive episode requires two weeks of four of the following symptoms in the face of depressed mood and/or anhedonia: *lowered interest in life, loss of appetite or weight loss, sleep disturbance, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or guilt, difficulty thinking or concentrating, morbid preoccupations.*

B. Though feelings of inappropriate guilt are mentioned, suicidal patients usually have this feeling to a marked degree. There is no mention of *anguish* as a feature of depression. It would be possible to see a patient who was filled with feelings of intense self-loathing, intense anguish, who felt utterly hopeless, and a serious, detailed suicide plan, and who was severely agitated. In the absence of other findings such a patient would not meet the criteria for a *DSM IV* diagnosis of depression.

C. Suicide is rare in schizophrenia, alcoholism, or in borderline personality disorder in the absence of depression marked by psychic anguish and self-loathing.

D. A comment on borderline personality disorder, commonly comorbid for depression.

V. What are the suicide risk factors to consider in affective disorders?

A. Those established by prospective empirical research (Fawcett, et al., 1990): Prospectively followed 954 patients with amajor affective disorders for 10 years and found nine clinical features associated with suicide:

1. Predictive of suicide within a year of admission: *panic attacks, severe psychic anxiety, diminshed concentration, global insomnia, moderate alcohol abuse,* and *severe loss of interest or pleasure (anhedonia).*

2. Predictive of suicide occurring after a year: *severe hopelessness, suicidal ideation,* and *history of previous suicide attempts.*

B. Risk factors supported by other research and by clinical experience: *Depressive illness or other Axis I. disorder; Alcoholism and drug abuse; Suicide ideation, talk, preparation; previous suicide attempts, especially involving lethal methods; isolation, living alone, loss of support; hopelessness; aging white male; modelling: suicide in the family, genetics; marital or family problems, including divorce; severe loss; anger, aggression, irritability (5-HIAA); physical illness, and combinations of any of the above.* --Maris, p. 9

C. Suicide in uncomplicated hypomania is unknown.

D. Suicide in "mixed states" (so-called manic dysphoria) is common.

VI. How to put it all together? "Formulation" of suicide risk. (Maltsberger, 1986)

A. History of intolerance of affect storms

B. Affects inviting suicide: rage, aloneness, self-hate, profound shame

C. Exterior supporting resources

D. Capacity to make use of resources

E. Reality testing

Suggested Readings

Akiskal, Hagop S.; Hirschfeld, M. A.; and Yerevanian, B. I. (1983) The Relationship of Personality to Affective Disorders. *Archives of General Psychiatry, 40:* 801-810

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Kraepelin, E. (1904) *Lectures on Clinical Psychiatry*. Translated and edited by Thomas Johnstone. London: Baillière, Tindall and Cox. (This book was reissued in facsimile in 1968 by the Hafner Publishing Co., New York.) ☞ See especially the lectures on melancholia, depressed stages of maniacal-depressive insanity, mixed conditions of maniacal-depressive insanity.

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Maris, Ronald W., Overview of the Study of Suicide Assessment and Prediction. Maris, R. W.; Berman, A. L.; Maltsberger, J. T.; and Yufit, R. I., Eds. (1992) *Assessment and Prediction of Suicide*. New York: The Guilford Press

Murphy, George (1992) *Suicide in Alcoholism*. New York: Oxford University Press

Robins, Eli (1981) *The Final Months*. New York: Oxford University Press

B. Barraclough et al. (West Sussex County, Great Britain, 1966–68, 100 suicides)[8]

Depressive illness	70%
Alcoholism	15%
Schizophrenia	3%
Phobic-anxiety state	3%
Barbiturate dependence	1%
Acute schizo-affective disorder	1%
Not mentally ill	7%

(Depressive illness + Alcoholism = 85%)

T.L. Dorpat and H.S. Ripley (Seattle, Washington, 1957–58, 108 suicides)[9]

Depressive illness	28%
Alcoholism	26%
Schizophrenia	11%
Personality and sociopathic disorders	9%
Organic brain syndrome	4%
Miscellaneous	3%
Unspecified psychiatric illness	15%
No psychological information	5%

(Depressive illness + Alcoholism = 54%)

E. Robins et al. (St. Louis, Missouri, 1956–57, 134 suicides)[10]

Affective disorder, depressed phase	47%
Alcoholism	25%
Organic brain syndrome	4%
Schizophrenia	2%
Drug dependence	1%
Undiagnosed psychiatric illness	15%
Terminal medical illness	4%
Well	2%

(Affective disorder, depressed phase + Alcoholism = 72%)

from Robins, E., p. xiv

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Department of Mental Health

Guidelines for Continuing Medical Education Credits (CME)

Sponsor: McLean Hospital

The Department of Mental Health has begun a series of new initiatives in Public Managed Care, and is committed to training its state and provider workforce to understand and field these new healthcare approaches. As part of this effort, DMH is underwriting the cost of today's program (including CME credits) because DMH believes in the importance of this topic. To obtain CME credits, registrants are required to remain for the entire training day. CME certificates will not be awarded until the end of the training day.

To ensure that the granting of CME certificates proceeds efficiently for all registrants, the following guidelines have been put in place:

1. All registrants must register and sign in for CMEs each training day in the registration area before 10:15 AM or other designated time. Any registrant who does not sign in by the designated time will forfeit CMEs for that training day. In case of a two-day or longer program, each registrant must sign in each day before the 10:15 AM or designated time.

2. All registrants must provide their Board of Registration license numbers before CMEs can be issues. These license numbers may be provided during the conference itself, or called in after the conference. In the latter case, DMH will mail the registrant the certificate.

3. Each registrant must hand in a completed course evaluation in order to receive a certificate.

4. CME credits are granted to physicians for each day of training at the end of the day's programming. Daily certificates may be obtained in the registration area at the end of the program.

These guidelines have been drawn up so that they are in accordance with all current licensing requirements for CMEs. The Department of Mental Health is responsible to its registrants and to its CME/CEU sponsors, and there are no exceptions to these Department of Mental Health requirements.

The designated registration time for CMEs for this program is no later than: 10:15am

Department of Mental Health

Guidelines for Continuing Education Credits (CEU)

Sponsor: Harvard Medical School

The Department of Mental Health has begun a series of new initiatives in Public Managed Care, and is committed to training its state and provider workforce to understand and field these new healthcare approaches. As part of that effort, DMH is underwriting the cost of today's program (including CEU credits) because DMH believes in the importance of this topic. To obtain CEU credits, registrants are required to remain for the entire training program. CEU certificates will not be awarded until the end of the training program.

To ensure that the granting of CEU certificates proceeds efficiently for all registrants, the following guidelines have been put into place:

1. All registrants must register and sign in for CEUs each training day in the registration area before 10:15 AM or other designated time. Any registrant who does not sign in by the designated time will forfeit CEUs for that program. In case of a two-day or longer program, each registrant must sign in each day before 10:15 AM or the designated time.

2. All registrants must provide their Board of Registration license numbers before CEUs can be issued. These license numbers may be provided during the conference itself, or called in after the conference. In the latter case, DMH will mail the registrant the certificate.

3. Each registrant must hand in a completed course evaluation in order to receive a certificate.

4. CEU credits are granted at the end of the program. The certificates may be obtained in the registration area.

These guidelines have been drawn up so that they are in accordance with all current licensing requirements for CEUs. The Department of Mental Health is responsible to its registrants and to its CME/CEU sponsors, and there are no exceptions to these Department of Mental Health requirements.

The designated registration time for CEUs for this program is no later than: 10:15am